



# Acute Cerebellitis: A Rare Complication of Multisystem Inflammatory Syndrome in Children (MIS-C)

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Received: 7 March 2022 / Accepted: 2 May 2022 / Published online: 16 July 2022  
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*To the Editor:* An 11-y-old male presented with high-grade fever, diarrhea, vomiting, multiple episodes of generalized tonic–clonic seizures, and altered sensorium. Examination revealed fever, heart rate 160/min, respiratory rate 24/min, well-palpable pulses, blood pressure 104/72 mm Hg, Glasgow Coma Scale of 10, pupils 3 mm equal size and reacting to light, decreased tone, normal deep tendon reflexes, extensor planter response, normal fundus examination, and no signs of meningeal irritation.

Investigations revealed anemia, thrombocytopenia, neutrophilic leucocytosis, lymphopenia, transaminitis, elevated inflammatory markers (CRP, procalcitonin, ferritin, and IL-6), deranged coagulogram, elevated D-dimer, and positive SARS-CoV-2 serology. Microscopic examination of the stool, stool and blood cultures, peripheral smear for malarial parasite, WIDAL test, and ELISA for scrub typhus were negative; and serum ammonia and cerebrospinal fluid (CSF) analysis were normal.

The initial management included intravenous ceftriaxone and antiseizure medications; and intravenous immunoglobulin (2 g/kg) and methylprednisolone (2 mg/kg/d) (for multisystem inflammatory syndrome in children, MIS-C). By day 3, there was improvement in fever, inflammatory markers, and encephalopathy, when he was noted to have cerebellar

signs (titubation, intention tremors, positive finger nose test, dysmetria, ataxic gait, dysdiadokokinesia, and dysarthria). Magnetic resonance imaging of the brain was normal. CSF autoimmune encephalitis panel (NMDA, AMPA, GABA<sub>A</sub>, GABA<sub>B</sub>, and CASPER antibodies) was negative. Possibility of acute cerebellitis in association with MIS-C was considered and the dose of methylprednisolone was escalated to 30 mg/kg/d (5 d). There was some improvement in cerebellar signs by day 8 and significant improvement by 2-mo follow-up.

Neurological involvement in MIS-C is documented in 25%–80% children in the form of headache, encephalopathy, meningism/meningitis, seizures, muscle weakness, and bulbar palsy; and signal changes in the splenium of the corpus callosum and acute demyelinating encephalomyelitis as common neuroimaging findings [1–3]. Neuroinflammation could be a possible reason for neurological manifestations in MIS-C. Acute cerebellitis or acute cerebellar ataxia is seldom reported in association with MIS-C [4].

## Declarations

**Conflict of Interest** None.

## References

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